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Relationships among Anxious Symptomatology, Anxiety Sensitivity and Laboratory Pain Responsivity in Children

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Abstract

Existing laboratory-based research in adult samples has suggested that anxiety sensitivity (AS) increases an individual's propensity to experience pain-related anxiety which in turn enhances pain responsivity. Such relationships have not been examined in younger populations. Thus, the present study used structural equation modeling (SEM) to test a conceptual model in which AS would evidence an indirect relationship with pain intensity via its contribution to state-specific anticipatory anxiety in relation to a variety of laboratory pain tasks (cold pressor, thermal heat, and pressure pain) in 234 healthy children (116 girls; mean age = 12.6 years, range = 8–18 years). The model further hypothesized that existing anxious symptomatology would demonstrate a direct relationship with pain intensity. Results of the SEM supported the proposed conceptual model with the total indirect effect of AS accounting for 29% of the variance in laboratory pain intensity via its effects on pain-related anticipatory anxiety. AS did not however, evidence a direct relationship with pain intensity. Anxious symptomatology on the other hand, demonstrated a significant direct effect on pain intensity, accounting for 15% of variance. The combined effects of AS, anxiety symptoms, and anticipatory anxiety together explained 62% of the variance in pain intensity. These relationships did not differ for boys and girls indicating no moderating effect of sex in the proposed model. The present results support the potential benefit of assessing both AS and anxiety symptoms in children prior to undergoing painful stimulation.

Keywords

children; adolescents; anxiety; anxiety sensitivity; laboratory pain; experimental pain; pain intensity

Introduction

There is growing support for a strong association between pain conditions and anxiety disorders in the general adult population (McWilliams *et al.*, 2004) and in adults with chronic medical conditions (Tsao *et al.*, 2004a). Although fewer studies have examined younger samples, there is evidence for a robust link between anxiety disorders and pain complaints in children (Garber *et al.*, 1990; Hodges *et al.*, 1985; Hyams *et al.*, 1996). Accordingly, a recent study demonstrated marked similarities in psychological symptoms and laboratory stress responsivity in children with recurrent abdominal pain (RAP) and children with anxiety disorders (Dorn *et al.*, 2003). Children with RAP and children with anxiety disorders evidenced similar levels of state anxiety in anticipation of a laboratory stress task despite higher existing levels of trait anxiety and anxious symptomatology in those with anxiety disorders relative to those with RAP. The authors concluded that although children with RAP may not be as likely to endorse increased

trait anxiety or anxiety symptoms as do children with clinical anxiety, both groups demonstrated similar levels of state anxiety and physiological arousal in response to a potentially threatening situation, in this case, a laboratory stressor.

Previously, we noted that state-specific ratings of anticipatory anxiety about impending procedures are proximal measures that can be conceptualized as indices of perceived aversiveness or threat (Tsao *et al.*, 2004b). These proximal measures of anxiety may be considered related to, though partially distinct from, more distal, trait measures of anxiety, which may also influence pain. Existing anxious symptomatology may constitute one marker of distal anxiety. Another potential marker is anxiety sensitivity (AS), or the specific tendency to interpret anxiety sensations as dangerous (Reiss *et al.*, 1986; Taylor, 1999). AS is a relatively stable dispositional construct that has been linked to anxiety disorders (Taylor, 1999), and chronic pain (Asmundson, 1999; Norton & Asmundson, 2004). Asmundson and colleagues proposed that AS may play a key role in the onset and maintenance of chronic pain by amplifying the tendency to develop fear of pain. The fear of pain in turn enhances pain-related avoidance, leading to deconditioning and increased pain resulting in further avoidance and negative expectancies regarding pain (Asmundson *et al.*, 1999). Although this model has yet to be studied in children with chronic pain, recent work has revealed elevated AS and anxious symptomatology in pediatric patients with noncardiac chest pain compared to controls (Lipsitz *et al.*, 2004). In healthy adolescents, AS has demonstrated a unique relationship to fear of pain, after controlling for pain and anxiety symptoms (Muris *et al.*, 2001b).

Laboratory pain studies in adults have generally shown robust relationships between AS and self-reported pain responses (e.g., pain intensity) (Keogh & Birkby, 1999; Keogh & Cochran, 2002; Keogh & Mansoor, 2001). Schmidt and Cook (1999) found that adults with panic disorder evidenced increased pain intensity to a laboratory pain task compared to healthy controls but that AS mediated the relationship between diagnostic status and pain intensity. In addition, they found that rather than directly influencing pain intensity, AS evidenced an indirect relationship to pain intensity via its contribution to pain-related anxiety. Previously, in a study conducted with a subset of children (N = 118) that took part in the current investigation, we found that state-specific anticipatory anxiety was an important predictor of laboratory pain responsivity (Tsao *et al.*, 2004b). AS did not predict incremental variance in pain responses after controlling for anticipatory anxiety and anxious symptomatology. However, in this earlier study, we used standard linear regression techniques and therefore could not specifically test the hypothesis that AS may have an indirect relationship with pain (i.e., via the influence of AS on anticipatory anxiety).

Thus, the present study used structural equation modeling (SEM) to test a conceptual model (see Figure 1) in which AS would predict pain-related anticipatory anxiety, which in turn would predict pain intensity in response to a variety of laboratory pain stimuli in a sample of healthy children. In light of previous findings of a significant relationship between anxious symptomatology and pain responses (Tsao *et al.*, 2004b), the model further posited that anxiety symptoms would directly predict pain intensity. We chose pain intensity as our measure of pain reactivity because extensive research in adults (Keogh & Birkby, 1999; Keogh & Mansoor, 2001; Schmidt & Cook, 1999) and in children (Tsao *et al.*, 2004b) has not generally shown reliable associations between AS and objective measures of laboratory pain response. Prior work in adults has also revealed important sex differences such that women with high AS exhibited elevated levels of sensory and affective pain (Keogh & Birkby, 1999; Keogh *et al.*, 2004; Keogh & Mansoor, 2001), but no such associations in men. We therefore explored the potential moderating effect of sex in our conceptual model.

Methods

Participants

Participants were 234 children (116 girls; 49.6%) (mean age: 12.6 years; $SD = 3.16$, range = 8–18). Participants were part of a larger study on puberty and pain response; the broad age range was designed to include the oldest age at which children were expected to be pre-pubertal, through adolescence. Ethnic composition was: 39.7% Caucasian, 14.1% African-American, 10.3% Asian-American, 23.1% Hispanic, 12.8% Other. Over 75% were middle to upper SES (Hollingshead, 1975). Recruitment procedures are detailed elsewhere (Lu *et al.*, 2005; Tsao *et al.*, 2004b). Briefly, participants were recruited through mass mailing, posted advertisements, and classroom presentations. Following confirmation of initial eligibility and verbal consent from a parent by telephone, written informed parental consent and child assent forms were mailed for review and signature. The UCLA Institutional Review Board approved all procedures. Participants received a \$30 gift certificate and a T-shirt for participation. In total, 244 healthy children (124 female) provided written informed consent. Of these, 10 had incomplete questionnaire data and were excluded; the final sample consisted of 234 children.

Procedure

Details of the laboratory procedure are provided elsewhere (Lu *et al.*, 2005; Tsao *et al.*, 2004b). In brief, on the day of the laboratory session, participants and their parents were greeted by an experimenter and escorted to separate rooms; there was no contact between them until after the session was finished. Participants first completed questionnaires in a quiet room. They were then led into the laboratory and instructed on the use of the visual analog scales (VAS) for rating pain and anticipatory anxiety (described below). Three practice trials were completed to ensure participants understood the VAS: 1) “How afraid or nervous would you be right before taking an important exam or test?” 2) “How much would it bother you to eat your favorite dessert?” 3) “How afraid, nervous or worried do you feel right now?” The instructions and practice trials were repeated until participants fully understood the VAS. Participants were then instructed about and exposed to the three pain tasks. Task order was counterbalanced across participants. Participants were not informed of the trial ceilings.

Cold Pressor Task—Participants placed the non-dominant hand to a depth of 2" above the wrist in 10 °C water in a commercial ice chest measuring 38 cm wide, 71 cm long and 35 cm deep. A plastic mesh screen separated crushed ice from a plastic large-hole mesh armrest in the cold water. Water was circulated by a pump to prevent local warming about the hand. The first trial had a ceiling of 3 minutes. Only the data from this trial were included herein. A second trial with a fixed ceiling of 1 minute was also performed using the dominant hand; these data are reported elsewhere (Tsao *et al.*, 2003).

Pressure Task—The Ugo Basile Analgesy-Meter 37215 (Ugo Basile Biological Research Apparatus, Comerio, Italy) was used to administer focal pressure through a lucite point approximately 1.5 mm in diameter to the second dorsal phalanx of the middle finger or index finger of each hand. Four trials at two levels of pressure (322.5g and 465g) were administered with a ceiling of 3 minutes.

Thermal Heat Task—The Ugo Basile 7360 Unit (Ugo Basile Biological Research Apparatus, Comerio, Italy) was used to administer four trials of two infrared intensities (15, 20) of radiant heat 2" proximal to the wrist and 3" distal to the elbow on both volar forearms, with a ceiling of 20 seconds.

Between each trial, there was a 1-minute inter-trial interval. For the thermal heat and pressure tasks, the presentation order (setting, site) was counterbalanced across participants. Before the

start of each trial, subjects were informed that they would experience moderate sensation, which may eventually be perceived as pain. Participants were instructed to continue with the task for as long as they could, and to withdraw from the apparatus if it became too uncomfortable/painful.

Measures

Questionnaires—The Childhood Anxiety Sensitivity Index (CASI) (Silverman *et al.*, 1991) is an 18-item scale assessing the tendency to view anxiety sensations as dangerous. Items are scored on a 3-point scale (none, some, a lot); total scores are calculated by summing all items. The CASI has evidenced good internal consistency ($\alpha = .87$) and adequate test-retest reliability over 2 weeks (range = .62 – .78) (Silverman *et al.*, 1991). The CASI correlates well with measures of trait anxiety (r 's = .55 – .69) but also accounts for variance in fear not attributable to trait anxiety measures (Weems *et al.*, 1998).

The Multidimensional Anxiety Scale for Children (MASC) (March, 1997) is a 39-item measure of anxiety symptoms comprised of four subscales representing empirically-derived domains of anxiety: physical symptoms, social anxiety, harm avoidance, and separation anxiety. Items are scored on a four-point scale (never, almost never, sometimes, often); total and subscale scores are calculated by summing the relevant items. The MASC has demonstrated high internal consistency ($\alpha = .88$ for total score) and adequate test-retest reliability over 3 months (March, 1997).

Pain Task Measures

Anticipatory Anxiety—Ratings of anticipatory anxiety were obtained immediately prior to each trial. Participants used a vertical sliding VAS, anchored with 0 at the bottom indicating the least amount and 10 at the top indicating the greatest amount, in response to the instruction to rate “how nervous, afraid, or worried are you about” the upcoming task. The scale also had color cues, graded from white at the bottom to dark red at the top, as well as a neutral face at the bottom and a negative facial expression at the top.

Pain Ratings—Immediately after each trial, participants were asked to rate the level of pain experienced during the trial using the same VAS as described above. For each trial, participants were asked, “at its worst, how much pain did you feel” during the trial.

Results

Descriptive Statistics

For the thermal heat and pressure tasks, pain intensity and anticipatory anxiety ratings were highly correlated across the four trials (r 's = .53 – .89, $p < .001$). Therefore, these data were averaged across the four trials yielding a mean thermal heat intensity rating, a mean pressure intensity rating, a mean thermal anticipatory anxiety rating, and a mean pressure anticipatory anxiety rating. Consistent with normative data (March, 1997), girls had higher MASC scores than boys (girls – $M = 42.48 \pm 13.64$; boys – $M = 38.04 \pm 13.25$, $t(232) = 2.53$, $p = 0.01$). Girls and boys did not differ on the CASI, nor were there sex differences in pain intensity and anticipatory anxiety for the three pain tasks. Bivariate correlations among the measured variables in the total sample are displayed in Table 1. All correlations were significant at $p = 0.01$. Although the impact of child age on the hypothesized relationships was initially considered for inclusion in our conceptual model, bivariate correlations among the measured variables did not change substantially after controlling for child age. Therefore, in order to test a more parsimonious model, age was not included in the final conceptual model.

Structural Equation Modeling Overview

EQS program version 6.1 (Bentler, 2003) was used to test the hypothesized structural equation model (SEM) using standard maximum likelihood (ML) estimation. For samples of $N \leq 250$, Hu and Bentler (1999) recommended combinational rules to evaluate model fit, with a value of 0.95 or above for the comparative fit index (CFI) and a value of or below 0.09 for the standardized root mean-square residual (SRMR) indicating good fit. In addition, Hu and Bentler recommended values of at least 0.95 for the non-normed fit index (NNFI), and 0.06 or below for the root mean-square error of approximation (RMSEA) for good model fit. ML estimation assumes multivariate normality. Multivariate kurtosis Mardia's coefficient was 14.77 and normalized estimate was 8.94, suggesting that the multivariate distributions were non-normal. Therefore, the Maximum-likelihood-robust method was used to estimate all models.

To evaluate model fit across multiple groups, SEM analyzes parameters simultaneously to determine which of several models best reproduces the sample data in each group. Starting with a baseline unrestricted model, increasingly restrictive hypotheses may be tested by constraining certain key parameters to equality across groups. If there is no significant difference in chi-square values between the models, the more constrained model is considered superior. If there is significant difference in chi-square values between models, the less constrained model is considered to fit significantly better than the more constrained model.

Confirmatory factor analysis

An initial confirmatory factor analysis (CFA) was performed in the total sample with each hypothesized latent construct (i.e., anticipatory anxiety and pain intensity) predicting its measured indicators. This analysis assessed the adequacy of the proposed factor structure (measurement model) and the relationships among the latent and measured variables. The model fit the data well, Satorra-Bentler Scaled χ^2 ($5, N = 234$) = 3.55, $p = 0.62$, CFI = 1.00, NNFI = 1.00, SRMR = 0.017, RMSEA = 0.00. Each path coefficient was statistically significant ($p < .05$). As expected, pain ratings for the three tasks reliably reflected an underlying latent construct of pain intensity (Cronbach's alpha = 0.80). The loadings (standardized path coefficients) for the cold, heat and pressure pain tasks on the latent factor of pain intensity were: 0.61, 0.84, and 0.84 respectively. Anticipatory anxiety ratings for the three tasks also reliably reflected an underlying latent construct of anticipatory anxiety (Cronbach's alpha = 0.82). Standardized path coefficients of cold, heat and pressure on the latent factor of anticipatory anxiety were 0.64, 0.86 and 0.85 respectively.

Structural Equation Modeling (SEM) and Multiple Group Comparison

Our model posited that CASI scores would predict anticipatory anxiety, which in turn would predict pain intensity (see Figure 1). The model also posited that MASC scores would directly predict pain intensity. We expected that MASC and CASI scores would be correlated and specified this in the model. We also expected that error residuals between the pain intensity and anticipatory anxiety for each pain task would be associated since both were measured using the VAS. Error residuals of the measured variables for anticipatory anxiety and pain intensity for each pain task were allowed to be freely estimated (e.g., residual of pressure pain intensity correlated with residual of pressure anticipatory anxiety). To test these hypotheses, analysis of the model was initially performed for the total sample. The model fit the data well, Satorra-Bentler Scaled χ^2 ($15, N = 234$) = 17.60, $p = 0.28$, CFI = 0.97, NNFI = 0.94, SRMR = 0.033, RMSEA = 0.027. Each path coefficient was statistically significant ($p < .05$) (Figure 1). The indirect effect of CASI on pain intensity via the anticipatory anxiety latent factor was 0.29. As shown in the Figure, the direct effect of anticipatory anxiety on pain intensity was 0.74 and the direct effect of anxiety symptoms on pain intensity was 0.15. Together, AS, anxious symptomatology, and anticipatory anxiety accounted for 62% of the variance in pain intensity.

To test the hypothesis that sex moderated the relationships among these variables, a series of analyses were conducted to determine whether girls and boys differed significantly on: (1) the factor loadings for latent pain intensity and latent anticipatory anxiety constructs, (2) the paths between CASI and anticipatory anxiety, and (3) the path between MASC and pain intensity. No differences were found between girls and boys; therefore, the hypothesized model for the whole sample was considered the best-fitting model.

Indirect Effects

The indirect effects of the MASC on pain outcomes via anticipatory anxiety and the direct effect of CASI were also examined. An alternative model was tested with additional path from MASC to anticipatory anxiety. The path was not significant, suggesting no indirect effect of MASC through anticipatory anxiety on pain intensity. An alternative model was also tested with additional path from CASI to pain intensity. The path was not significant, suggesting no direct effect of CASI on pain.

Discussion

Our results support the proposed conceptual model (Figure 1) in that AS was found to predict pain-related anticipatory anxiety which in turn predicted laboratory pain intensity in this sample of healthy children. In total AS indirectly explained 29% of the variance in pain intensity via its effects on anticipatory anxiety. Consistent with the hypothesized model, AS did not evidence a direct effect on pain intensity. The observed indirect relationship between AS and laboratory pain response is consistent with the view, proposed by Schmidt and Cook (1999), that AS enhances pain intensity by increasing an individual's vulnerability to experiencing anxiety, which then in turn promotes increased pain. Also consistent with our proposed model, anxious symptomatology was found to demonstrate a direct relationship with pain intensity, accounting for 15% of the variance. On the other hand, anxiety symptoms did not evidence a clear link with pain-related anticipatory anxiety. Whereas this latter finding may seem counter-intuitive, our measure of anxiety symptoms (the MASC) assessed four different domains of anxiety (i.e., social anxiety, separation anxiety, harm avoidance, and physical symptoms); not all of these domains would be expected to show strong associations with state-specific anxiety in anticipation of painful stimulation. The observed distinction between anxious symptomatology and state-specific anxiety is consistent with the work of Dorn et al. (2003) who found that although children with anxiety disorders endorsed higher levels of anxiety symptoms than children with RAP, the groups did not differ on state anxiety prior to a laboratory stress task. Thus, the present findings suggest that AS and anxious symptomatology represent related but partially distinct constructs that independently influence laboratory pain responses directly, in the case of anxiety symptoms, and indirectly in the case of AS, via pain-related anticipatory anxiety.

As mentioned above, our results agree with those of Schmidt and Cook (1999) who found that AS in adults was linked to increased pain report via heightened anxiety in response to the cold pressor task. Most prior work examining the relationship between AS and laboratory pain has been conducted using a single type of pain stimuli (i.e., the cold pressor task) in adult samples. In addition, few existing studies have distinguished between the potential influence of distal, trait measures of anxiety (e.g., AS; anxious symptomatology) and proximal measures of anxiety (e.g., anticipatory anxiety) in relation to pain response. We were able to test the proposed conceptual model across three different tasks employing cold, thermal heat and pressure pain stimuli. Our analyses also confirmed that pain intensity responses as well as anticipatory anxiety ratings for the three pain tasks each reliably reflected underlying latent factors of pain intensity and anticipatory anxiety, respectively. These findings suggest that self-reported pain intensity was unified across the different types of pain stimulation, as was

anticipatory anxiety. Moreover, the observed effects for AS and anxious symptomatology held for children's responsivity to all three pain tasks. Our conceptual model also revealed a small but statistically significant direct link between anxiety symptoms and the latent factor representing pain intensity. These findings are consistent with earlier work showing an association between anxiety symptoms and pain in adults with panic disorder (Schmidt *et al.*, 2002) and high rates of comorbidity between chronic pain and anxiety disorders in adults (Asmundson & Taylor, 1996). Dorn *et al.* (2003) similarly found 50% of their sample of children with RAP met criteria for lifetime anxiety disorder.

Our findings were somewhat at odds with existing studies in adult samples indicating significant sex differences in AS-pain relationships. Research by Keogh and colleagues (Keogh & Birkby, 1999; Keogh *et al.*, 2004; Keogh & Mansoor, 2001) has shown that the association between AS and pain holds mainly in adult women but not in adult men. In the present study however, we did not find a moderating effect of sex within the proposed conceptual model. Boys and girls did not differ on the factor loadings for the latent pain intensity and latent anticipatory anxiety constructs, nor were there any differences between boys and girls in the relationships among AS, anticipatory anxiety, anxious symptomatology and pain intensity. One possible reason for the divergence in findings is the wide age range in the present sample which was chosen to include the oldest age at which children were expected to be pre-pubertal though adolescence. It may be that responses among older adolescents would more closely mirror those of adults. Thus, there may be potential interactions between sex and age which were not specifically examined in the current study. For the sake of parsimony, we did not include the potential influence of age in our current hypothesized model. As mentioned above, correlations among the measured variables did not change substantially after controlling for age. Future work may compare AS-pain relationships in older adolescent vs. pre-adolescent samples in addition to age by sex interaction effects.

The present findings are consistent with the conceptualization of AS as a key cognitive vulnerability factor in the subjective experience of pain. Thus, Greenberg and Burns (2003) found evidence supporting the notion that in adult chronic pain patients, "pain anxiety" is best conceptualized as an expression of AS or the disposition to fear anxiety-provoking stimuli in general, rather than a circumscribed phobia of painful stimuli. As several authors have pointed out (Schmidt & Cook, 1999; Watt & Stewart, 2000; Watt *et al.*, 1998), anxiety sensitivity may be viewed as the propensity to perceive any source of arousal as threatening and thus, AS may amplify the experience of bodily symptoms related to a wide range of somatic events. Thus, Muris *et al.* (2001a) found significant associations between parental transmission of the idea that somatic symptoms may be dangerous and AS levels in healthy adolescents. They noted that parental transmission was associated with the experience of both anxiety and pain symptoms suggesting that such learning experiences likely involve parental confirmation of sick-role behavior related to bodily symptoms in general. In accord, Watt and colleagues (1998) have proposed that elevated AS may develop as a consequence of learning to catastrophize about somatic sensations generally rather than anxiety symptoms specifically. Despite the potential influence of parental AS beliefs on children's pain responding, few studies have examined the impact of parent AS on children's experimental and "real world" pain (e.g., injections). Such studies in younger healthy and chronic pain samples are warranted.

Limitations to the present study should be mentioned. The current findings are cross-sectional and no conclusions regarding causality may be inferred. Although the results suggest that our conceptual model fit the data closely, this does not rule out other possible causal models. Prior work supports the hierarchical structure of the CASI (Silverman *et al.*, 2003)—however, our sample size was insufficient to allow the inclusion of the CASI or the MASC as latent factors. The strong association between our latent constructs of pain intensity and anticipatory anxiety may be partly due to shared method variance as the same VAS was used to measure these

responses. Other studies however, including Schmidt and Cook (1999), have also used the same VAS scale to assess both pain and anxiety ratings in relation to laboratory pain tasks. A related limitation is the possibility that there may have been a greater overlap between the concepts of pain and anxiety among younger children due to lower levels cognitive development (e.g., less ability to distinguish between pain and anxiety). Nevertheless, we did include three practice trials to ensure that the VAS was understood by all participants. It should be noted that whereas these practice trials did include a rating of state anxiety, they did not include a pain rating.

Earlier studies in pediatric samples have shown that anticipatory anxiety predicts post-operative pain ratings (Palermo & Drotar, 1996) and pain reactions to bone marrow aspiration (Hilgard & LeBaron, 1982), but the potential role of AS and anxious symptomatology in relation to such procedures has not been assessed. Our results suggest that interventions designed to target AS may lead to reductions in anticipatory anxiety which may in turn have beneficial effects on children's responses to acute pain. The observed direct effect of anxiety symptoms on pain intensity also suggests that children with elevated levels of anxious symptomatology may be more vulnerable to experiencing increased pain. The identification and targeting of this subgroup using treatments that have been found to be effective in clinical anxiety may also lead to reductions in pain reactivity (Dorn et al., 2003). Future study on the associations among proximal state-specific and distal, trait factors related to anxiety and how they influence pain response may assist in the development of more effective interventions for pain in children.

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Figure 1.

Conceptual model with standardized path coefficients and factor loadings illustrating the relationships among anxiety symptoms (MASC), anxiety sensitivity (CASI), anticipatory anxiety and pain intensity. Latent constructs are in circles, indicators are in boxes; 1-headed arrows depict standardized regression paths, 2-headed arrows represent correlations (standardized covariances).

Table 1

Bivariate Correlations among the Measured Variables

	CASI	MASC	Cold anticipatory anxiety	Pressure anticipatory anxiety	Thermal heat anticipatory anxiety	Cold pain intensity	Pressure pain intensity rating
MASC	0.54						
Cold anticipatory anxiety	0.32	0.19					
Pressure anticipatory anxiety	0.31	0.22	0.55				
Thermal heat anticipatory anxiety	0.34	0.26	0.53	0.73			
Cold pain intensity	0.21	0.26		0.35	0.39		
Pressure pain intensity	0.25	0.21	0.46	0.73	0.55	0.51	
Thermal heat pain intensity	0.30	0.34	0.44	0.56	0.73	0.53	0.70

Note. All of the above correlations were significant at $p = 0.01$ (two tailed)